

Bull (C.S.)

ON SOME

# FORMS OF KERATO-IRITIS

AND THEIR

*EFFECT IN HINDERING THE OSMOTIC ACTION  
OF THE CORNEA AND CONJUNCTIVA.*

BY

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[REPRINTED FROM THE NEW YORK MEDICAL JOURNAL, SEPT., 1874.]

NEW YORK:

D. APPLETON AND COMPANY,

549 & 551 BROADWAY.

1874.

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(Established May, 1872.)

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*Presented by  
A. E. M. Purdy*

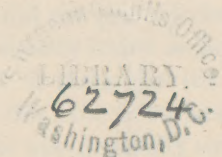
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## ON SOME FORMS OF KERATO-IRITIS AND THEIR EFFECT IN HINDERING THE OS- MOTIC ACTION OF THE CORNEA AND CONJUNCTIVA.

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OPHTHALMOLOGISTS not uncommonly meet with cases of kerato-iritis, in which atropine, applied locally, seems to produce little or no beneficial effect in allaying the inflammatory symptoms, and in which the pupil dilates but little or not at all, though there may be no posterior synechiæ present. Why is it that, in some cases of corneal inflammation, atropine acts promptly and beneficially, while in others it is apparently an inert agent? Is there a *peculiar* kind of corneal or iritic inflammation, in which atropine is useless as a remedial agent, and is this form of inflammation marked by any one symptom or chain of symptoms, which may be regarded as pathognomonic?

The cases in which the writer has observed this peculiarity were all chronic, and did not present themselves for treatment until the disease had already lasted several weeks, and in some cases months. In all of them the iris was more or less affected, the nature of the iritic inflammation being generally serous. The keratitis was generally somewhat diffuse, sometimes circumscribed, always parenchymatous, and the corneal epithelium was generally wanting over a greater or less extent of surface. Sometimes the cornea seems to be the seat of a regular abscess, which is afterward converted into an



open ulcer by a portion of necrotic corneal tissue sloughing away. The iris is always discolored and generally swollen, but yet does not present the bagging, bulging appearance which accompanies complete posterior synechiæ, with effusion into the posterior chamber. There are no vessels visible in the cornea proper, except at the periphery, where we meet with the loops of the fine branches of the anterior ciliary arteries going to the conjunctiva. These patients have remained under treatment for weeks at a time without any improvement being visible, and have then suddenly taken a turn for the better. They are not distinguished by any one symptom or chain of symptoms from other cases of parenchymatous keratitis, except by their obstinacy in resisting treatment. They are accompanied by little or no pain, and by but little photophobia. The keratitis does not seem to be a manifestation of the strumous diathesis, nor of any other particular constitutional dyscrasia, but is apparently a purely local trouble.

Is the inertness of the atropine due to its not being absorbed, or to some other reason? If it is not absorbed, is it because the corneal inflammation has so changed the cornea that it has lost the properties of an animal membrane, or is it owing to some defect or obstruction in the circulation; and, if the latter, can the sympathetic nerve be brought in as a factor in the case?

When the coats of the eye are in the normal condition, we know that a solution of atropia is absorbed directly by the corneal tissue as well as by passing into the circulation through the conjunctival vessels. In considering the osmotic action of the cornea, we must regard it as an animal membrane. We know that *one* condition of the penetrability of porous bodies by liquids, or their power of absorption, is the faculty of becoming moistened, or the attraction which is set up between a molecule of fluid and the walls of a pore, which, in the case of the cornea, means a lymphatic space; another condition is the attraction which one molecule of liquid has for another. We also know that the molecules of liquid, having once entered an animal membrane, are retained in the pores of the membrane by the same two conditions of attraction existing between the walls of a pore and the different

molecules of fluid, and by the cohesion acting between different molecules of the same fluid. Another circumstance to be taken into consideration is, that the state of humidity or moisture of the animal substance and its absorbent power for liquids exercise a certain influence upon the rate of transmission of a fluid through its tissues. Of all fluids, distilled water is absorbed in the largest proportion, and the power of absorption of saline solutions diminishes as their concentration increases.

Now, in order that a solution of atropine may transude the cornea and pass into the anterior chamber, some of the aqueous humor must drain off from the anterior chamber, otherwise there must be an increase of intra-ocular tension, which, if maintained, would lead to grave trouble. This may or may not pass through the cornea outward, though experiments of somewhat recent date would lead us to believe that the aqueous humor never transudes the corneal tissue, no matter how great the intra-ocular tension may be which is brought to bear upon the cornea. If these experiments, however, be not true, there is a mingling of the aqueous humor and the solution of the atropine as well in the tissue of the cornea as in the anterior chamber, which takes place in the pores or the lymphatic spaces of the cornea. The connection between a membrane and the rate of its osmotic action we know to be very intimate, and, in those cases of keratitis where atropine acts much less promptly than in others, this is probably due, in great part, to the condition of the cornea. In inflammation all the physical laws of health and nutrition are interfered with, and the tissue inflamed, and every thing that takes place in and around it must be regarded as morbid and abnormal. It is natural to suppose that the well-known laws governing osmotic action are interfered with in such a case. The chemical combination or union of two liquids, separated by an animal membrane, only takes place when the force of attraction is stronger than all the obstacles against which it has to contend. When the membrane is healthy, it exercises no direct influence upon the osmotic current, if the temperature remain the same throughout the experiment.

If we agree to regard the cornea as filled with hollow, very



narrow spaces, the lymphatic spaces, and admit that the solution of atropia passes through and meets in the substance of the cornea with the aqueous humor, there ought to be just as strong a current in one direction as in the other, always premising that the corneal tissue is healthy. When the cornea is the seat of the inflammation, the power of absorption may vary in different portions of the membrane. In keratitis, the cornea is very often thickened, which of itself would alter its osmotic power, for it is a well-known fact that the rapidity of the two currents of endosmosis and exosmosis depends upon the thickness of the membrane separating the liquids. Some supposed that, when the parenchyma of the cornea is inflamed, the membrane of Descemet, or rather the single layer of hexagonal cells upon the posterior surface of the cornea, exerts a great influence upon the absorptive power of that membrane, but this is as yet a mere hypothesis in the pathology of the cornea.

But the cornea is not the only tissue concerned in the absorption of atropine. The blood-vessels of the conjunctiva are to be considered under this head, and perhaps play a much more important part in the process than the cornea itself. They possess one condition which is very favorable to absorption, viz., within them is contained a fluid which circulates, which moves at a certain rate, a condition which physiology teaches us is more favorable for promoting the osmotic action than when the two liquids are quiet. Here the wall of the blood-vessel represents the membrane, while the fluids are represented by the blood on one side and the solution of atropine on the other. This motion of the blood in the capillaries favors imbibition by removing that portion of the fluid which has already been absorbed and carrying it forward in the circulation.

The question may now be asked, What influence the state of the sympathetic nerve exerts upon the osmotic or absorptive power of the blood-vessels of the conjunctiva. We know, from Broussais, that the great sympathetic gives off throughout its course three kinds of branches; the muscular, the visceral, and the vascular. The latter are sent to the arterial and capillary branches which supply blood to the different organs of



the body, embrace the vessels, and form plexuses around their coats. These arteries are furnished with nerve-twigs as often as they pass by a ganglion or a plexus, while the veins and lymphatics do not receive any nerve-supply from the sympathetic nerve. Now, in inflammation of a part, the arteries of the part involved dilate, become thicker and larger, and pulsate more strongly, while, on the subsidence of the inflammation, they revert to their original size and state. All this is a manifestation of organic sympathy due to the action of the sympathetic nerve. Now, if the vessels are dilated and pulsate more strongly, the blood must course through them more rapidly, and thus favor the process of imbibition. Hence we may conclude that a solution of atropia is not only absorbed more rapidly in such a case, but more of it is absorbed, and that the characteristic effect upon the pupil ought to be produced so much the more rapidly, provided that there are no posterior synechiæ present. But in the class of cases under consideration the pupil does not dilate, and hence we must look yet farther for a satisfactory explanation. We now turn our attention to the iris, and consider it with special reference to its muscular action and nerve-supply. We know that the muscular tissue of the iris is of the non-striated variety, and is arranged in two different ways, with a more or less intimate connection between them. Some of the fibres are arranged in a circular direction around the margin of the pupil, forming what is called a sphincter muscle of the iris, and these fibres run very closely together. Other fibres run from the external margin of the sphincter muscle in a radiating direction toward the periphery of the iris, but are arranged in bundles or fasciculi, each fasciculus being separated by a greater or less distance from the neighboring ones, the space being occupied by elastic tissue. We know that the diminution in size of the pupil is a double act, consisting of a relaxation of the radiating muscular fibres and a contraction of the circular fibres or sphincter muscle, while in dilatation of the pupil the reverse is the case.

We know that the nerves of the iris are branches of the ciliary nerves, and consist of three sets, viz.: 1. Pale fibres, probably belonging to the sympathetic nerve, which run tow-

ard the posterior surface of the iris and ramify *probably* in the dilatator iridis or radiating muscle. 2. Fibres which run on the anterior surface of the iris, which are its sensory fibres. 3. Motor fibres running to and in the sphincter muscle (Iwanoff, Stricker's "Gewebelehre," page 1047). In other words, the ciliary nerves supply motor, sensory, and sympathetic fibres to the iris. They all arise, with the exception of one or two branches which come directly from the naso-ciliary nerve, from the ciliary ganglion, which is situated on the external side of the optic nerve, about three-quarters of an inch behind its entrance into the globe. This ganglion has three distinct roots: *one*, the short or motor root, from the oculomotorius or third pair; *another*, the long or sensory root, from the naso-ciliary nerve, which is a branch of the ophthalmic branch of the trigeminus or fifth pair; *the third*, some fibres of the sympathetic, coming from the carotid plexus out of the cavernous sinus, through the superior orbital fissure, and generally reaching the ciliary ganglion itself, though sometimes it is joined with the long or sensory root before the latter's union with the ganglion.

It is well known that the normal movements of the iris occur in consequence of the irritation of the retina by the light, the transmission of this irritation from the retina, through the medium of the optic nerve, to the brain, and the reflex action of the oculomotorius. If the optic nerve be divided within the skull, the pupil dilates and remains so permanently. Division of the oculomotorius produces the same effect. Irritation of the motor oculi causes contraction of the pupil, and irritation of the sensory branches of the trigeminus gives the same result. Division of the trigeminus simply destroys the sensibility of the parts supplied by it, but does not disturb the relation existing between the optic nerve and the oculomotorius. A permanent contraction of the pupil has been observed in animals when the sympathetic nerve has been divided in the region of the third or fourth cervical vertebrae, and this depends upon the fact that the radiating fibres are paralyzed. A contraction of the pupil therefore follows, either in consequence of an increased action of the oculomotorius and the sphincter muscle, or in consequence of a weakened influence of the sympathetic nerve upon the radiating fibres.



Dilatation of the pupil follows in consequence of the weakened influence of the oculomotorius upon the sphincter, or in consequence of the increased influence of the sympathetic nerve upon the dilatator pupillæ.

Paralysis of the oculomotorius or of the ciliary nerves always causes dilatation and immobility of the pupil, that is, mydriasis. Atropine paralyzes the ciliary nerves, and irritates the dilatator pupillæ.

Now, it has been known for many years that division or injury of the fifth pair of cranial nerves within the cavity of the skull, or of its ophthalmic branch, is followed by an inflammation of the eye of the same side, which usually progresses to complete destruction of the organ. Immediately after the operation or injury, the pupil contracts, and the conjunctiva and cornea lose their sensibility. At the end of a varying period, the cornea becomes slightly cloudy, and, by the second day, the conjunctiva is already inflamed. The inflammation increases in intensity, and soon spreads to the iris, which becomes covered with a layer of inflammatory exudation. The cornea constantly grows more opaque, and may eventually ulcerate, and the contents of the anterior chamber, and even the lens and vitreous humor, may be evacuated, and the eyeball then collapses. We learn from Brown-Séquard that the same thing occurs after section of the lateral half of the spinal cord.

Now, in this form of trigeminal neuralgia or inflammation, whichever it may be, the effect produced upon the cornea is invariable and almost characteristic. The cornea is, as it were, paralyzed, cut off from its nerve supply, and the process almost invariably results in destruction of the cornea, unless one plan of treatment be resorted to, and even then we do not always succeed in staying the process of disintegration. The plan consists in closing the eye by a bandage, which is kept on continuously, and only removed to admit of an examination of the eye, the object being to protect the cornea from the injurious influences of light and air, and floating dust. Now, although the symptoms and course of this necrotic corneal process are very different from those of the form of keratitis considered in this paper, yet there is some slight resem-

blance between them, and, the affection of the trigeminus being closely connected with the corneal degeneration in the former cases, I am inclined to think that there is a closer connection between the same nerve and that form of corneal disease which opposes the local effect of atropine. Ophthalmologists observe every day cases of keratitis and kerato-iritis, in which the opacity and thickening of the cornea are very marked, and yet the pupil dilates readily under atropine, showing that the osmotic power of the cornea still exists. Even in old cases of corneal leucoma, where all signs of vascularity have long since disappeared, the pupil dilates easily and rapidly when atropine is instilled. Hence, in the class of cases under consideration, I am inclined to think that the sympathetic twigs to the iris play an important part in hindering the dilatation of the pupil by atropine, and that, though the atropine does pass through the cornea into the anterior chamber, or into the general circulation by means of the conjunctival and corneal vessels, yet once there, it fails in its specific action on the pupil, owing to some abnormal condition of the sympathetic nerve. What this condition is, whether an inflammation or an irritation, and where localized, whether in the iris or farther back, I am unable to say. It is also possible that the suspected diseased condition of the sympathetic exerts some influence upon the blood-vessels, thus retarding or even hindering the absorption of the atropia. It is very much to be hoped that some elucidation of this question may soon be furnished us, and, if this paper may be the means of calling the attention of my fellow-laborers in the field of ophthalmology more closely to the subject, I shall be more than satisfied.



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Professors H. SAINT-CLAIRE DEVILLE, BERTHELOT, and WURTZ, have engaged to write, but have not yet announced their subjects. Other eminent authors, as WALLACE, HELMHOLTZ, PABKS, MILNE-EDWARDS, and HAECKEL, have given strong encouragement that they will also take part in the enterprise.

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